A Review of Wound Healing and Wound Dressing Products

A brief review of the literature concerning the wound healing process is presented. A synopsis of the physical and physiologic factors that can affect the rate of this process is provided. The authors discuss the importance of the wound dressing in maintaining an optimal environment for wound healing. Modern dressings have specific indications and are frequently comprised of more than one layer. The authors define the contact layer as the layer that comes into intimate contact with the wound surface and has the greatest influence on healing. A description of some of the common products used as the contact layer is presented. (The Journal of Foot and Ankle Surgery 36(1):2–14, 1997)

Key words: wound dressings, wound healing, tissue healing

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The amount of knowledge and understanding concerning the wound healing process and dressing practices has expanded and changed dramatically over the past three decades. Prior to this time, the healing process was considered to be a passive process with respect to the physician (1–3). This theory was summed up by Ambroise Pare, who said "I dressed the wound, God heals it" (4).

Starting with the work of Winter and others in the early 1960s, much has been learned about the cellular and biomechanical components of wound healing and the factors that affect them (1, 2, 5). It is now known that healing is not a passive process, but rather can be accelerated and enhanced by the use of specific wound care/dressing techniques and products (1).

Stages of Wound Healing

After an injury or surgical procedure, the healing process begins. This consists of three phases and is found in all normal wound healing (6–8). These phases are not distinct, but form a continuum of the wound healing process (1, 3, 6, 8, 9).

The inflammatory or substrate phase begins immediately after wounding and lasts approximately 4 days (6, 8). The initial goal of this phase is hemostasis (6, 8). This is carried out through smooth muscle contraction and subsequent occlusion of the larger damaged blood vessels (1, 10). The activation and aggregation of platelets and release of clotting factors at the vessel wall injury site starts the coagulation process that helps to thrombose the smaller damaged vessels (1, 8, 10, 11).

A second goal of the inflammatory phase is the removal of bacteria, foreign debris, and other contaminants (6, 8, 10). Neutrophils, also known as polymorphonuclear leukocytes (PMNs), migrate from the surrounding microvasculature and serve to accomplish this. Although they appear first, PMNs are short-lived—lasting about 6 hr. (1).

Macrophages appear at the wound site approximately 48 hr. after injury. These cells, in addition to aggressively removing necrotic or foreign debris and phagocytizing bacteria, initiate two important aspects of healing—angiogenesis and fibroplasia (1–3, 11–13). These are mediated by various proteins or cytokines released by activated macrophages. Angiogenesis and the formation of capillary buds start at 3 days post-wounding and are essential for providing the metabolic needs of the healing process (1, 10, 13). Fibroplasia and collagen synthesis start by the third to fifth day post-injury (13).

The proliferative or fibroblastic phase begins toward the end of the inflammatory phase and lasts as long as 3 weeks (approximately 3 to 21 days post-injury) (1, 10). Activated macrophages already present in the wound site release angiogenesis factor (AGF) and fibroblast-stimulating factor as noted previously. AGF initiates the development of capillary buds and neovasculature. Fibroblast-stimulating factor cause the activation of fibroblasts at approximately 1 week after injury. Fibroblasts, thus, become the key cells of the proliferative phase (1). Collagen and proteoglycans are produced by the fibro-

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blast. The neovasculature, along with collagen and the proteoglycan ground substance, form granulation tissue. Granulation tissue fills in wound defects during the proliferative phase (1, 14). The production of collagen causes an increase in wound tensile strength between days 5 and 15 post-injury (1, 6). Contraction of the wound helps to reduce the size of the defect and brings the wound edges closer together. This process occurs between the first and second week after injury and is mediated by special fibroblasts with contractile properties called myofibroblasts (14).

The process of re-epithelialization occurs in the proliferative phase (3, 10, 14). This involves the proliferation and migration of epithelial cells over the granulation tissue to bridge the defect. Reepithelialization is essential to wound healing as the new epithelial cells provide a barrier to microbial invasion and help to prevent fluid loss (10). This step occurs rapidly in a wound closed by primary intention, but requires a longer time in wounds having a larger defect (10, 14). Because the epithelial cells must migrate over the wound surface, the rapidity of the migration is greatly affected by the type of surface. A moist wound surface facilitates epithelial migration, whereas a dry or eschar covered surface provides an impediment to migration (3, 10). As will be discussed later, the goal of many new wound dressing products is to provide a moist environment to enhance re-epithelialization.

The final phase of wound healing is called the maturation or remodeling phase. This begins at approximately 21 days post-injury and continues until 1 to 2 years after the injury (1, 6, 15). Early in this phase, fibroblasts continue to produce collagen (1, 6, 15). Remodeling is carried out by collagenases that are secreted during this phase (1). The collagen bundles synthesized during the proliferative phase and randomly laid are partially lysed by these enzymes. This allows for a debulking of the collagen as well as a reorganization of the bundles into a more parallel arrangement. Wound contraction and a gradual increase in tensile strength are seen as this phase progresses (1, 3, 6, 15). The ultimate strength of the healed wound is determined by the amount of collagen synthesis and the extent to which cross-linking has occurred between collagen bundles. Maximal tensile strength is achieved at the end of the maturation phase and is usually about 80% of the original, uninjured tissue's strength (10). There is some loss of elasticity seen in tissue that has undergone normal healing and, in some instances, this can cause adhe-

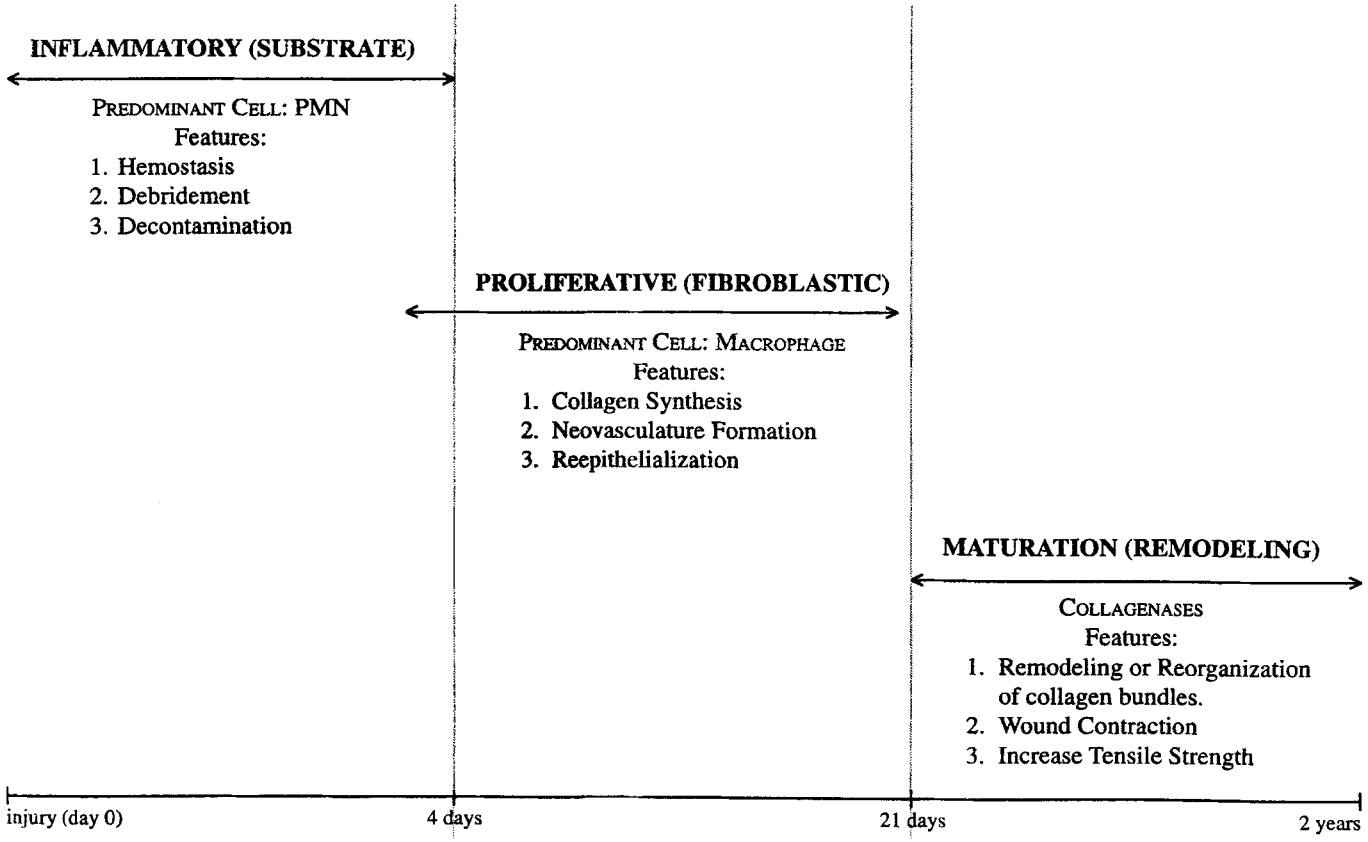

FIGURE 1 Timeline of normal wound healing phases.

sions or excessive contractures (1). The phases of wound healing are summarized in Figure 1.

Factors Affecting Wound Healing

It is well known that there are certain requirements for proper wound healing. Of paramount importance is adequate blood supply and tissue perfusion (1, 6, 10, 15). A surgical or traumatic wound with adequate perfusion has the needed influx of white cells for debridement and decontamination (10). Protein that is brought into the wound site is needed for collagen synthesis (10). Collagen synthesis, angiogenesis, and the phagocytic activity of the white blood cells are all dependent on oxygen tension (6, 10, 15). It has been shown that an increased oxygen tension at the wound site increases the healing rate while decreasing the incidence of infection (2, 10).

Several systemic conditions are known to affect the tissue perfusion of wounds. These include peripheral vascular disease of various etiologies, diabetes mellitus and aging, among others (1, 6, 7, 15–18). Various environmental stressors can reduce tissue perfusion (2). Excessive pain, cold, hypovolemia, or anxiety perceived by the patient can cause local vasoconstriction and increased healing time (2, 10). The components of tobacco smoke, namely nicotine and carbon monoxide, are known to decrease wound tissue perfusion and oxygen tension (10, 19).

Nutrition is of great importance in wound healing. It is accepted that a serum albumin level of 3.5 gm/dl or higher is necessary for proper healing. A malnutrition state may provide an inadequate amount of protein and this can manifest as a decreased rate of collagen synthesis, decreased wound tensile strength or an increased chance for infection (2, 6, 18, 20).

Patients with altered immune function will likely have an increased susceptibility to wound infection. The presence of an infection will adversely affect angiogenesis, the formation of granulation tissue and epithelialization (1, 17, 18, 20, 21).

Some pharmacological agents have untoward effects that can inhibit or disrupt the healing process (6, 18, 22, 23). The drug classes that most commonly affect the healing process are nonsteroidal anti-inflammatory drugs (NSAIDs), anticoagulants, and glucocorticoids (6). These agents can slow healing by exerting a negative influence on the initial inflammatory phase (18, 24). Hemostasis can be delayed by the action of NSAIDs and anticoagulants (6, 24). Glucocorticoids or steroids not only interfere with the migration of leukocytes to the wound site, but they can decrease their capacity for phagocytosis and intracellular killing (6, 18). In addition to delaying healing, the limited presence and efficacy of the initial leukocytes makes the wound site more susceptible to infection (18).

TABLE 1 Desired effects of dressing materials on wound environment

- Allow exchange of O_2 and CO_2
- Maintain moist environment
  - Avoid maceration
  - Avoid desiccation
- Permit transfer of drainage/exudate to 2° layer
- Protect site from physical injury
- Prevent wound contamination
- Prevent disruption of incision site (clean wounds closed by 1° intention)
- Aid in debridement (contaminated wounds—2° intention)

Some commonly used topical preparations may also negatively affect the rate of healing. Antiseptic solutions such as povidone-iodine, hydrogen peroxide, sodium hypochlorite solution, etc. are known to be cytotoxic, especially when not diluted (1, 2, 6, 8, 22, 23, 25). For this reason, these solutions should be diluted with a physiological solution such as normal saline or Lactated Ringer's solution if they must be used (2). It has been suggested that irrigation of the wound with only a physiologic solution is the most effective and least harmful method of wound cleaning (25). There are other systemic and topical medications that may have an effect

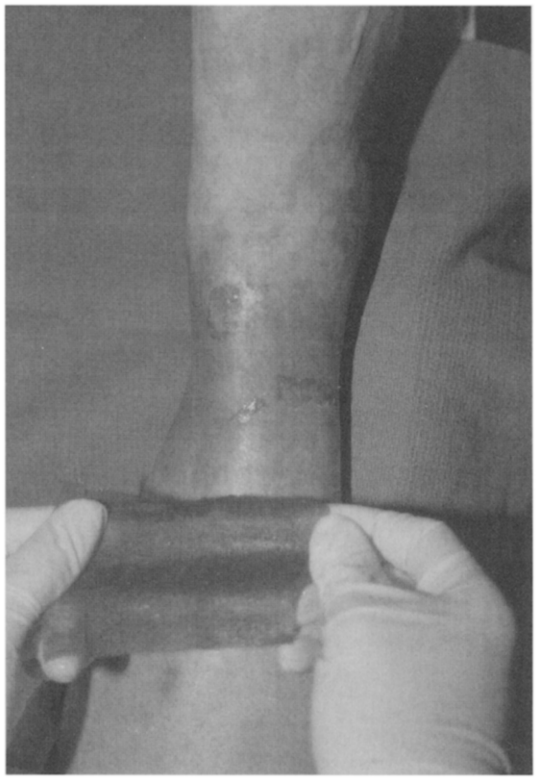

FIGURE 2 A common contact layer dressing, Betadine™ antiseptic gauze.

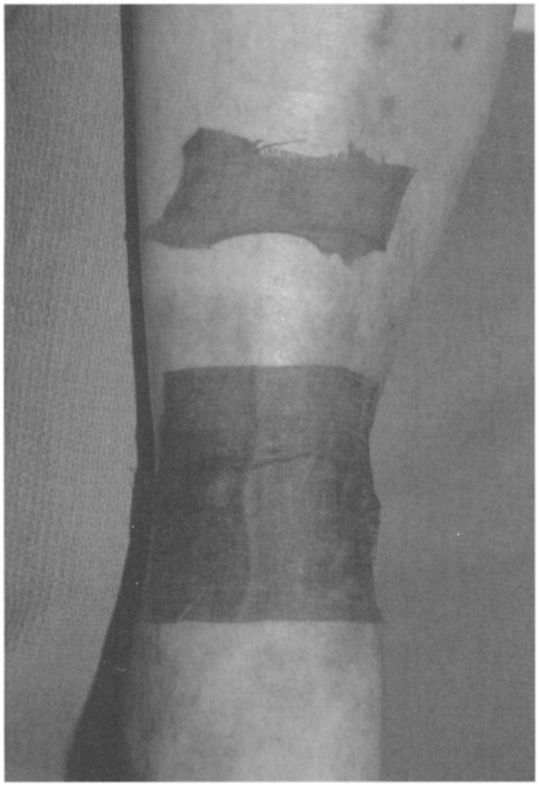

FIGURE 3 Betadine™ antiseptic gauze applied over superficial abrasions.

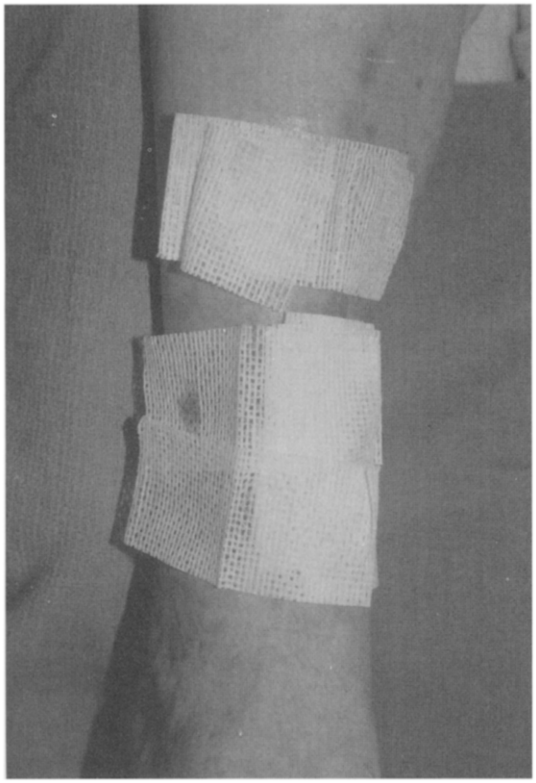

FIGURE 4 Secondary dressing, gauze applied to aid in absorption of exudate and act as padding.

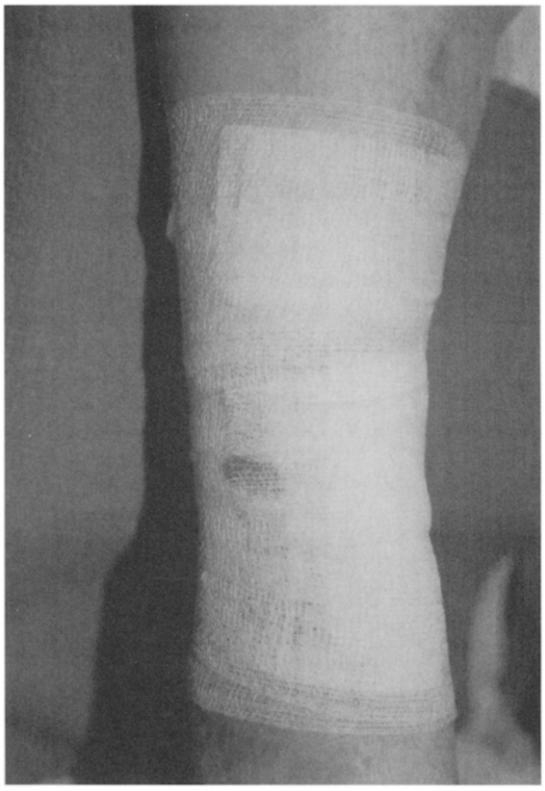

FIGURE 5 Secondary dressing, roll gauze.

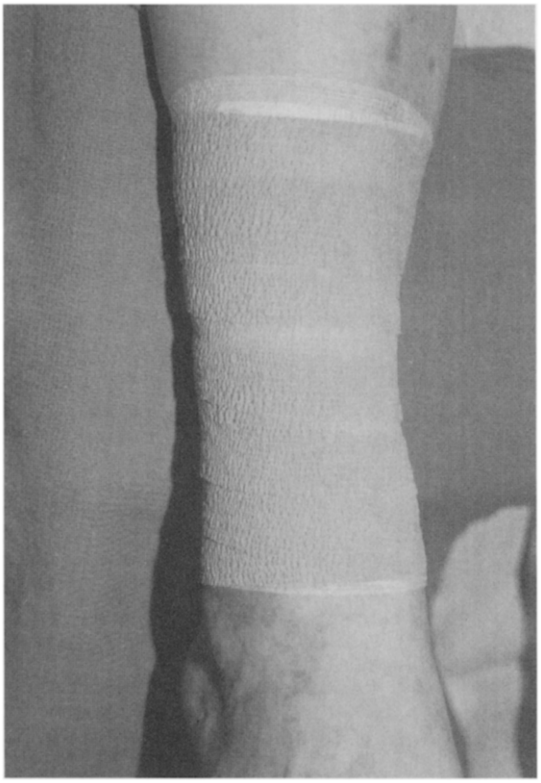

FIGURE 6 Secondary dressing, compressive layer.

on wound healing, however, only the most common have been presented.

Certain local conditions favoring wound healing are established and maintained by dressings (26). Prior to initial dressing application, foreign matter, necrotic tissue, and contaminating microorganisms should be removed as these predispose the wound to infection (26, 27). This can be carried out with careful attention to sterile, atraumatic surgical technique, or in the case of a traumatic wound, thorough initial debridement and irrigation (15, 26, 28). The dressing will then protect the wound site from further contamination (26, 28, 29). Maceration of the surrounding skin can be prevented by the absorption of excess wound exudate by the dressing (15, 26, 28–30). Re-epithelialization and cellular migration occur best in a moist environment, thus desiccation should similarly be avoided (2, 18, 24, 26). The dressing used should also be gas permeable—allowing for the introduction of oxygen to the wound site as well as elimination of carbon dioxide (15). The incidence of hematoma or abscess formation can be reduced by avoiding wound "dead spaces" through the use of compressive or packing dressings (26, 27). In some instances, the dressing can serve to debride the wound at dressing changes by adhering to necrotic tissue, dried exudate, etc. (15, 28–30). The effects of dressings on wound environment are shown in Table 1.

Wound Dressings

The contact layer of a wound or surgical dressing, as defined by the authors, is the layer that is placed directly over the wound prior to application of the rest of the dressing. The authors define the remaining layers as the secondary dressing. The secondary dressing generally consists of fluffs or gauze padding, roll gauze, and an outer, compressive layer (Figs. 2–6). The contact layer is vitally important because it can radically alter the characteristics of the dressing (2, 18, 27). Turner, et al have stated that "there is no single dressing that can produce the optimum microenvironment for all wounds or for [all] the healing stages of one wound" (31). Thus, the product or material used for the contact layer should be chosen based on the type of wound being dressed. The wound must be evaluated regularly for changes in size, depth, amount of exudate, presence or absence of infection or necrotic tissue, condition of the surrounding skin, and comfort level of the patient (2).

Classes of Contact Layer Dressings

Contact layer dressings can be divided into different classes based on their composition, indications and contraindications (2, 18, 26). Transparent film dressings are made of a self-adhesive polyurethane film (15, 29, 30). Examples of this type of dressing include Bioclusive™[3] and POLYSKIN I™.[4] These dressings are semipermeable, allowing gas and water vapor exchange, but preventing the absorption of wound exudates (26). Because they are occlusive to fluids, they should be used on drier wounds where they may help to provide a more moist environment and avoid desiccation (26). By preventing fluid loss, Transparent film dressings can help promote autolysis of necrotic tissue (2, 32). Maceration may result if a film dressing is used on wounds with even a moderate amount of drainage (26, 32). The thin polyurethane film clings and conforms well to the surface of the wound so they may be helpful in reducing friction or shear at the site (26). Further protection can be afforded by the use of a secondary dressing (padding, etc.). Transparent film dressings are also useful for abrasions, partial and full thickness wounds and intravenous catheter sites (15, 29, 30).

Hydrogel dressings come in the form of a gelatinous sheet or an amorphous wound filler (15, 26, 29, 30). They conform well to wound surfaces and can be used to fill wound defects (26). Hydrogels are nonadhesive and are permeable to gases. They provide little absorption, so they should be used on dry or lightly exudative wounds to maintain a moist surface. Their use on moderate to heavily draining wounds may result in maceration (26). A secondary dressing can be used to enhance absorption and to provide compression, however, these dressings can be used alone (15, 29, 30). NU-GEL™[5] is an example of the hydrogel class.

Probably the most commonly used dressings for surgical incisions or traumatic wounds closed by primary intention are the impregnated gauzes (26). These typically consist of a fine or open mesh gauze that is impregnated with a petrolatum emulsion or similar substance (26, 29, 30). The petrolatum allows these dressings to be nonadherent, thereby increasing patient comfort and preventing disruption of the incision site at dressing changes (26). Adaptic™[6] and Aquaphor Gauze™[7] are impregnated gauzes that are nonocclusive because of the mesh design. They can be used on moderate to heavily draining wounds including surgical incisions, abrasions and lacerations (26). When used in conjunction with an absorptive secondary layer, they can help prevent maceration of these exudative wounds (26). The transfer of exudate to the outer layer may aid in the prevention of infection (29, 30) (Figs. 7–9).

Petrolatum gauze U.S.P.[8] unlike Adaptic™ and Aquaphor™ gauze, is occlusive to fluids. This dressing is made

[3] Johnson & Johnson Medical Inc., Arlington, TX 76004-0130.
[4] Kendall Healthcare Products Company, Mansfield, MA 02048.
[5,6] Johnson & Johnson Medical Inc., Arlington, TX 76004-0130.
[7] Beiersdorf Medical Inc., Norwalk, CT 06856-5529.
[8,9] Sparta Surgical Corp., Hayward, CA 94545.

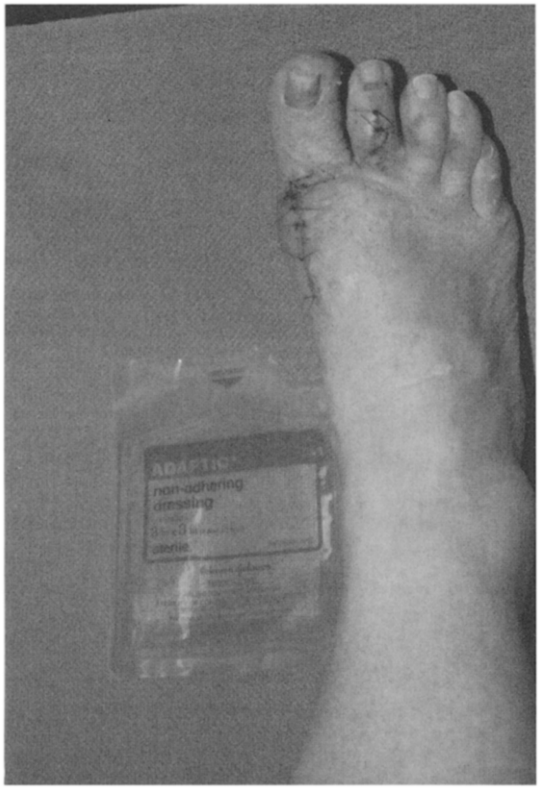

FIGURE 7 Adaptic used postsurgically to provide nonadherence.

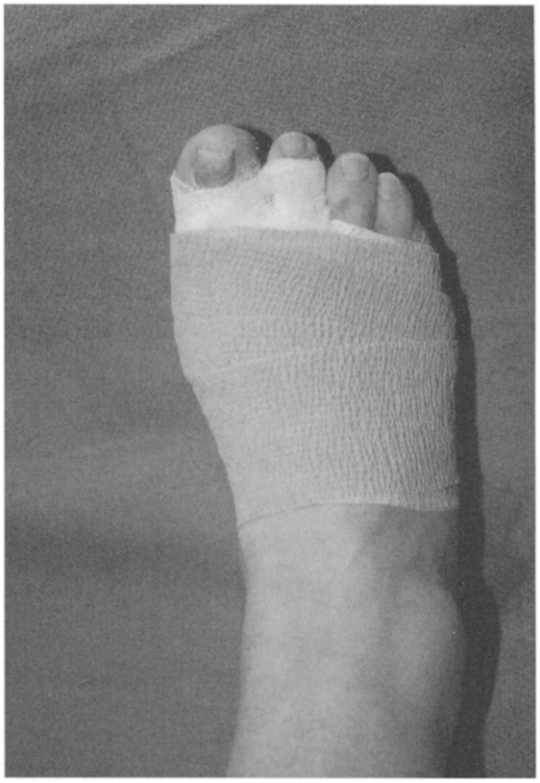

FIGURE 9 Standard secondary layer—gauze, roll gauze, and compressive layer.

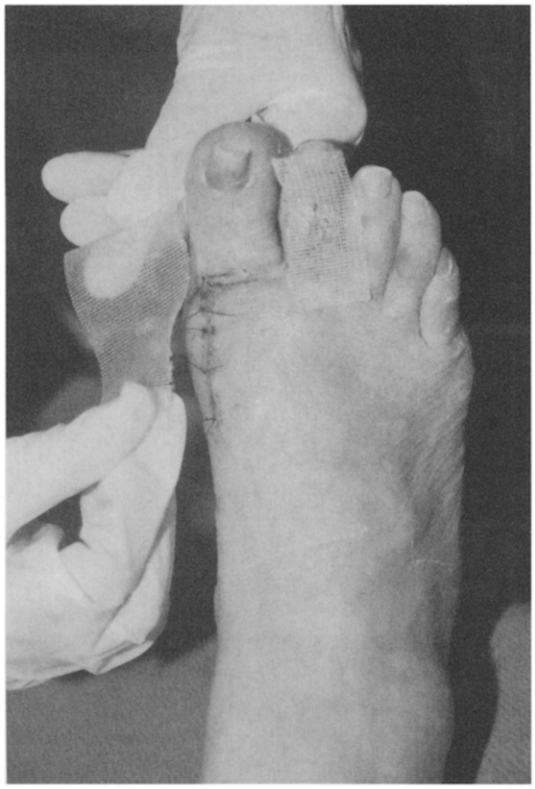

FIGURE 8 Adaptic applied over sutures.

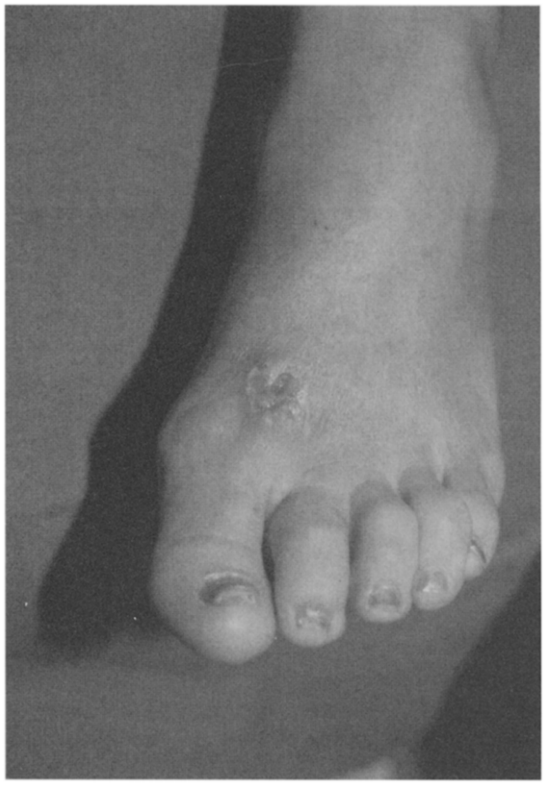

FIGURE 10 Open skin lesion with superficial infection.

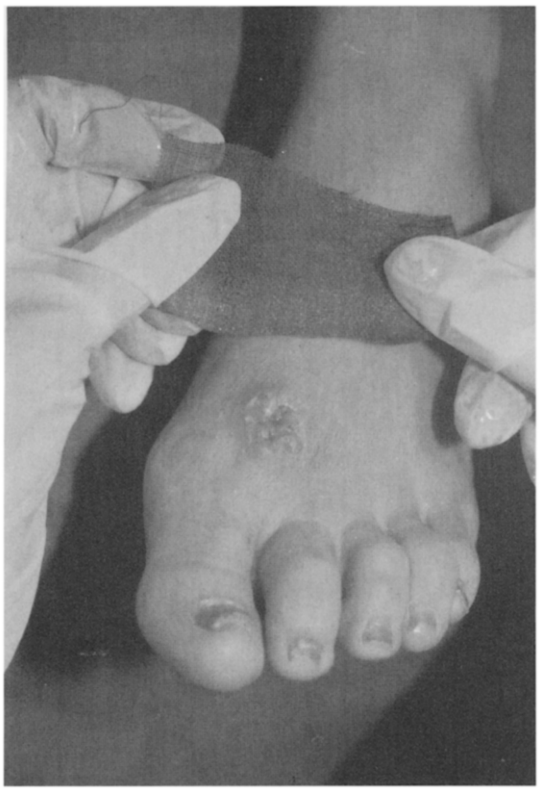

FIGURE 11 Betadine™ antiseptic gauze is selected for antimicrobial activity.

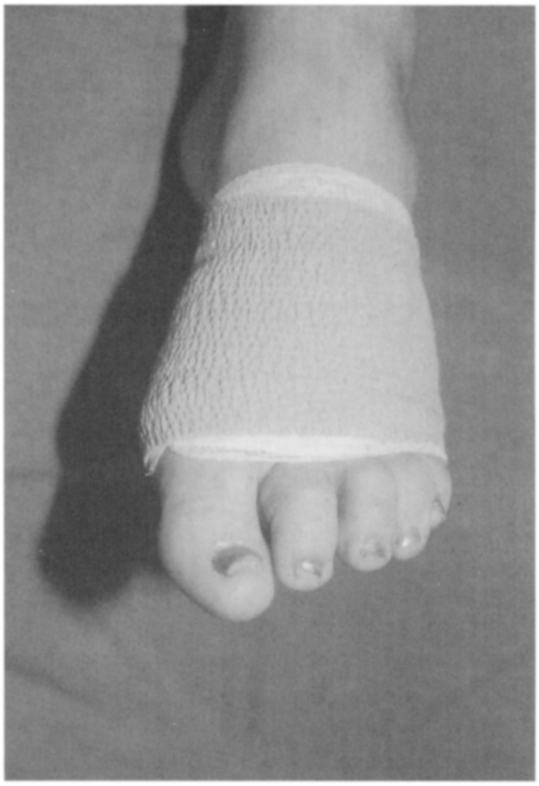

FIGURE 13 Secondary dressing is added to protect wound and prevent further contamination.

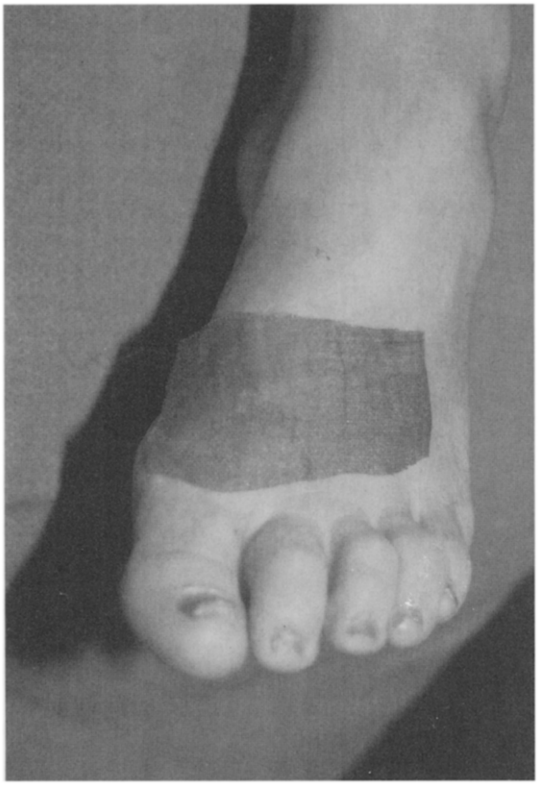

FIGURE 12 Contact layer is applied.

of a finer mesh that has a heavier coating of petrolatum (29, 30). It has its primary use on drier wounds, where the transfer of any available fluid would cause desiccation of the wound. Its use on draining wounds could result in maceration (29, 30).

Xeroform™[9] is also an occlusive dressing with essentially the same properties as petrolatum gauze U.S.P. Xeroform™ has, in addition to petrolatum, 3% Bismuth Tribromophenate, which acts as a mild astringent and deodorizer to further prevent fluid loss (29, 30). Xeroform™ should also be used on wounds that have little or no drainage, or where fluid loss needs to be prevented to ensure a moist wound environment (29, 30). These applications include surgical incisions, open wounds, graft sites, and burns.

Betadine™[10] antiseptic gauze is another impregnated gauze dressing. It is a regular mesh gauze that is impregnated with 10% povidone-iodine solution instead of petrolatum. This affords the dressing a bactericidal/virucidal nature, and it finds its greatest use in wounds that are contaminated or superficially infected (33, 34). This dressing should be avoided in patients with a history of allergy or hypersensitivity to povidone-iodine (33). The clinician should also be aware of the potential

[10] The Purdue Frederick Company, Norwalk, CT 06856.

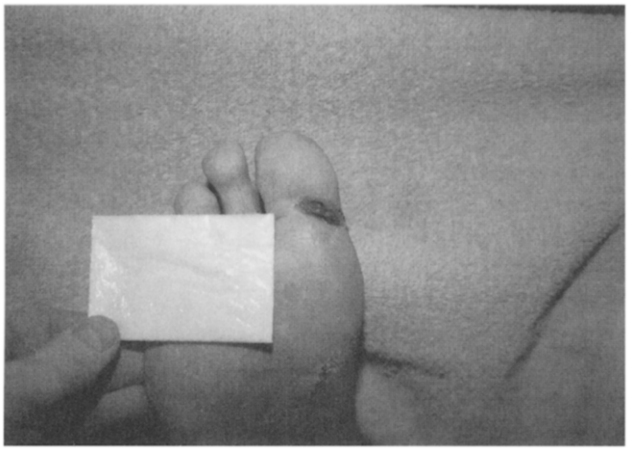

FIGURE 14 Telfa™ used as contact layer over lightly exudative ulcer.

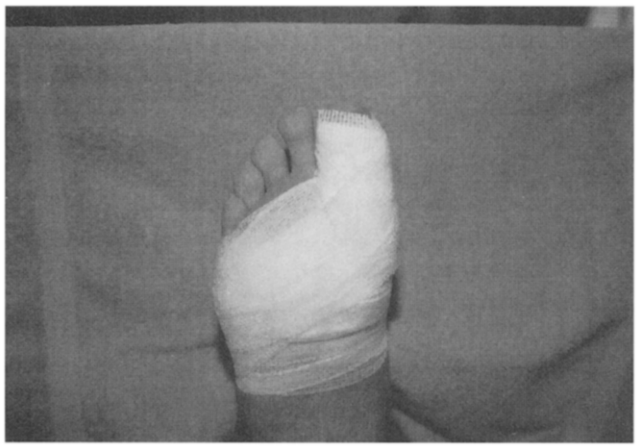

FIGURE 15 Secondary dressing applied over Telfa™.

cytotoxicity of povidone-iodine to new, fragile, or granulating tissue (33). In the authors' experience, this dressing is slightly more adherent to the wound site than other impregnated gauze dressings and tends to become stiff as it dries. This may cause a decrease in patient comfort or some disruption of the wound site at dressing changes (Figs. 10–13).

The next class of contact layer dressings are called nonadherent dressings. They are generally composed of two or more thin layers, including a porous inner layer that allows for the passage of exudates, and an outer, absorbent layer (29, 30). Although they are used as a nonadherent contact layer, they are slightly more adherent than the impregnated gauzes (26). Their absorptive nature makes them ideal for lightly exudative wounds such as surgical incisions, abrasions, ulcers, etc., where maceration is a potential problem (26) (Figs. 14, 15). In some instances they can be used as the secondary dressing to cover an impregnated gauze or other contact layer dressing (26, 29, 30). Examples of this type of dressing include Release™[11] and Telfa™.[12]

Wet dressings can be used in areas of dry skin such as seen with surgical incisions, burns, ulcers, etc. They can be used to moisten a dry wound or to provide wet to dry debridement (29). They consist of a gauze dressing that is saturated with either sterile water or saline solution (26). Sterile water[13] and isotonic saline[14] wet dressings are used to moisten and protect drier wounds from desiccation (29). Hypertonic saline[15] wet dressings, when combined with an absorbant secondary dressing, can effectively "draw out" exudates and contaminants from a wound while helping to reduce edema. Its use should be discontinued when the wound is no longer draining to prevent excessive drying of the wound site (29, 30). Owen's™ dressing[16] is a plain fine mesh that must be moistened with saline, sterile water, etc. prior to its application. Because of its finer mesh, Owen's™ dressing is not an effective contact layer when wet-to-dry debridement is desired, and should be used in situations where nonadherence is needed (29).

Hydrocolloids, such as Tegasorb™,[17] are occlusive dressings comprised of two layers. The inner layer is a wafer of a gum-like substance—usually pectin or karaya—that adheres to the wound surface. The outer layer is a water-resistant film or foam that allows patients to bathe with the dressing in place (26, 35). Hydrocolloids adhere and conform well to the wound surface and do not require a secondary dressing (26, 35). Wound exudate interacts with the inner wafer and forms a gel-like substance. The gel then helps to keep the wound surface moist and provides protection

[11] Johnson & Johnson Medical Inc., Arlington, TX 76004-0130.
[12] Kendall Healthcare Products Company, Mansfield, MA 02048.
[13–15] Sparta Surgical Corp., Hayward, CA 94545.
[16] American Cyanamid Co., Danbury, CT 06810.
[17] 3M Inc., St. Paul, MN 55144.

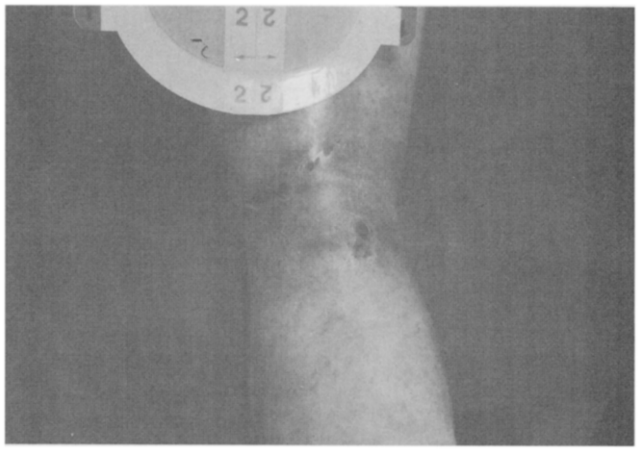

FIGURE 16 Tegasorb™ selected as contact layer over dry, slowly healing wound with some eschar (autolytic debridement needed).

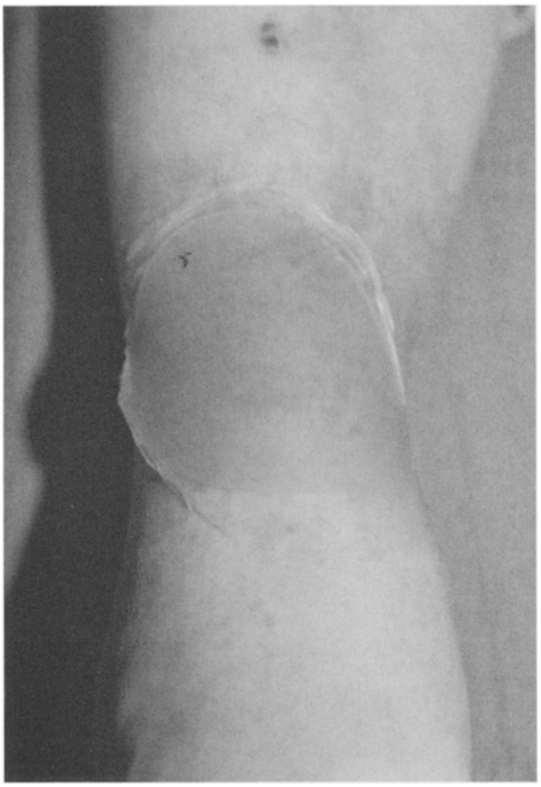

FIGURE 17 Application of Tegasorb™.

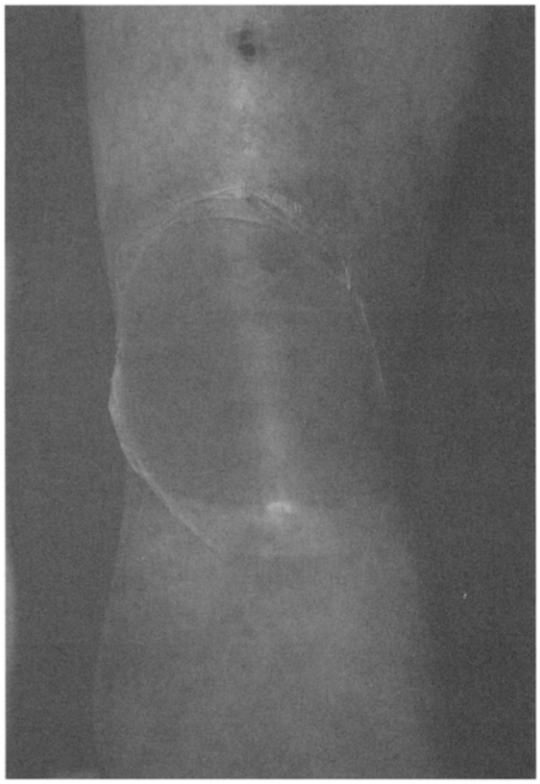

FIGURE 18 No secondary layer is needed with hydrocolloids.

against trauma and bacterial invasion (26, 35). These dressings can absorb a slight to moderate amount of exudate before maceration becomes a potential problem (35). The occlusive nature of these dressings makes them ideal for use on wounds where autolytic debridement of necrotic tissue is desired (32, 35). They should be avoided, however, on clinically infected wounds or for patients with increased risk of infection (35). Hydrocolloid dressings require changing every 1 to 7 days, depending on the type of wound and the amount of exudate present (35). Because of the relative infrequency of dressing changes, Hydrocolloids are generally more cost effective than other types of dressings such as wet to dry saline gauze (27, 36, 37) (Figs. 16–18).

Calcium alginate is a relatively new dressing material that is made of a naturally occurring polysaccharide

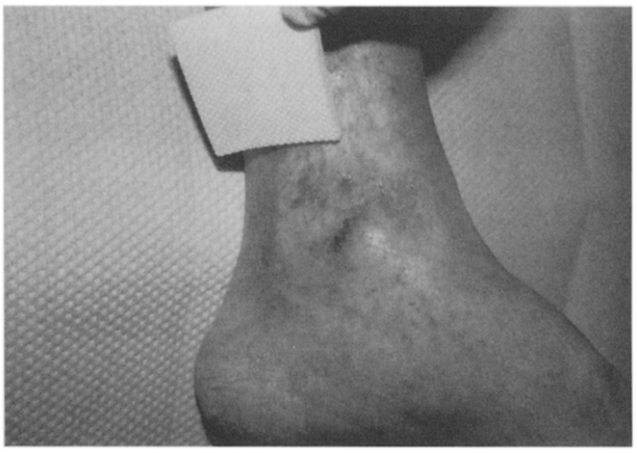

FIGURE 19 Composite dressing applied over lightly exudative venous stasis ulceration.

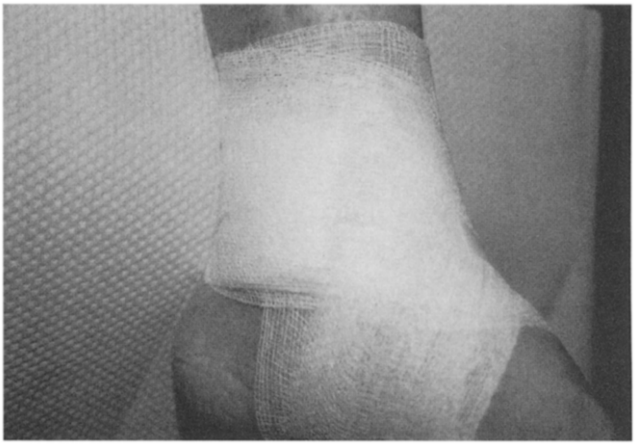

FIGURE 20 Secondary layer applied over composite dressing.

found in seaweed (26). It is a fibrous-appearing material that conforms to the wound surface. It may also be used as a packing material for wound defects or dead spaces (26). The dressing turns into a gelatinous mass as it absorbs exudate (26). This not only protects against maceration, but also helps to remove contaminates by wicking away or drawing out excess exudate from heavily draining wounds (26). The gelatinous mass protects the wound from physical trauma and increases patient comfort at dressing changes. The mass is simply flushed away with sterile irrigating solution prior to application of the new dressing. Calcium alginates do require a secondary dressing to help absorb exudate and to protect the gelatinous mass (26). Sorbsan™[18] is an example of a calcium alginate dressing.

The exudate absorbers make up a large category of dressings. They include some dressings that have already been discussed, including calcium alginates and Hypertonic Saline Gauze, as well as a variety of other materials including hydrolysate powder, starches, etc. (26, 29). In general, they conform well to wound surfaces and dead spaces where they can absorb up to 20 times their weight in exudate (26). As the name indicates, exudate absorbers are used for moderate to heavy draining wounds and can enhance autolytic debridement of necrotic tissue

[18] Dow B. Hickam Inc., Sugar Land, TX 77487.

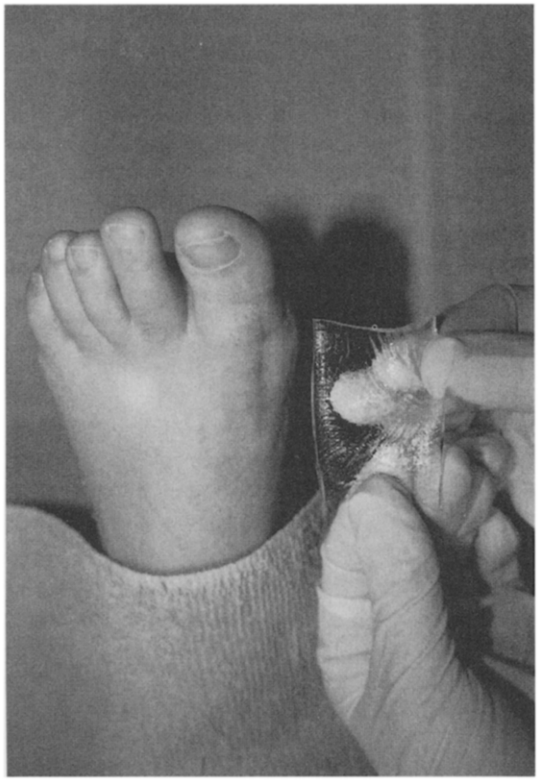

FIGURE 21 Moderately hypertrophic postsurgical scar.

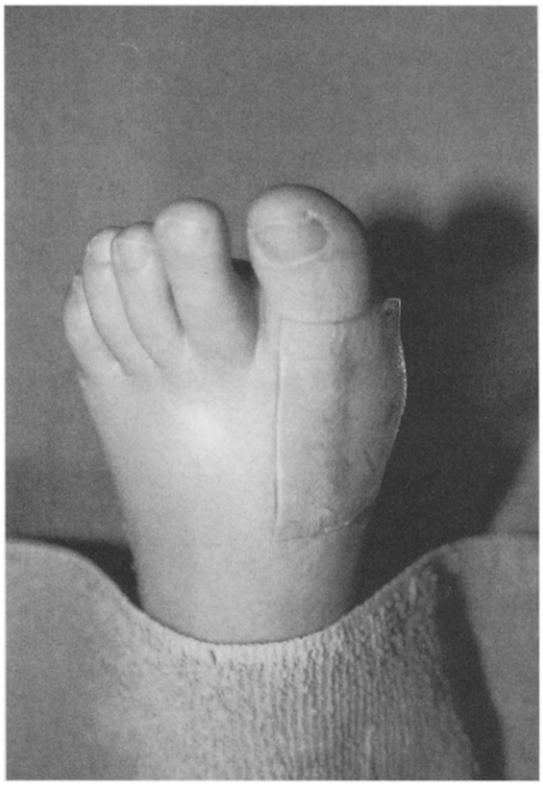

FIGURE 22 SILASTIC™ gel sheeting placed over scar.

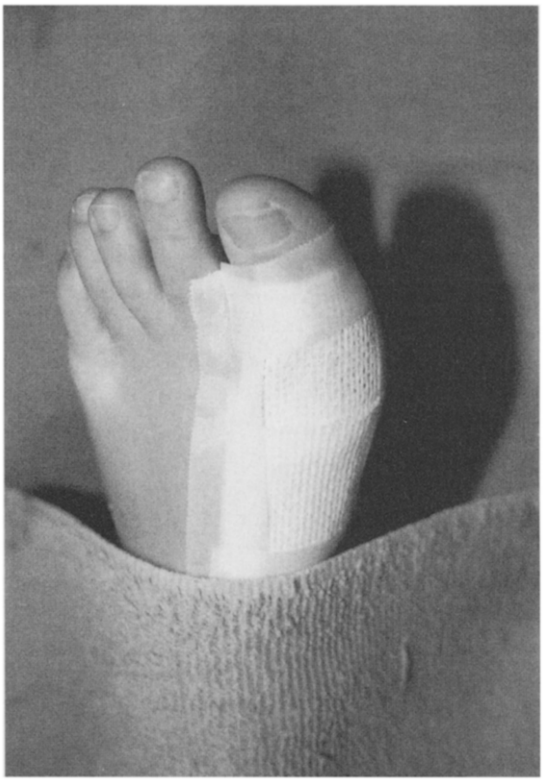

FIGURE 23 Secondary layer applied over SILASTIC™ gel sheeting.

(26). Because of their absorptive capacity, these materials are used with a secondary dressing. An example from this class is Debrisan™.[19]

Composite dressings are prefabricated combinations of dressing materials designed to meet a specific need. An example is Mitraflex™,[20] which combines a permeable, nonadherent contact layer, an absorptive middle layer, and an outer layer of semipermeable film to allow gas exchange (26). Dressings from this category may help to reduce the time needed for dressing changes while simplifying dressing selection (26) (Figs. 19, 20).

Patients who have a propensity for forming hypertrophic scars or keloids present a special problem. Silicone gel was first used to reduce burn scars in 1982 (38). Prior to this, treatment included topical emollients, cortisone injections, surgical revision, and various physical therapy modalities such as massage and ultrasound (38).

Dockery, *et al.* have very recently shown the benefits

[19] Johnson & Johnson Medical Inc., Arlington, TX 76004-0130.
[20] Calgon Vestal Laboratories, St. Louis, MO 63166.

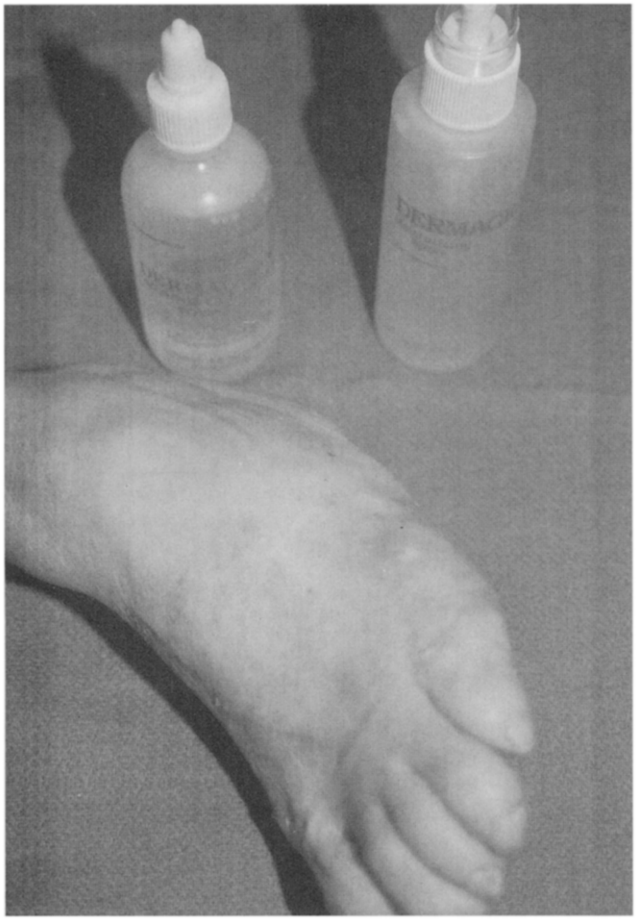

FIGURE 24 Slowly healing ulcer.

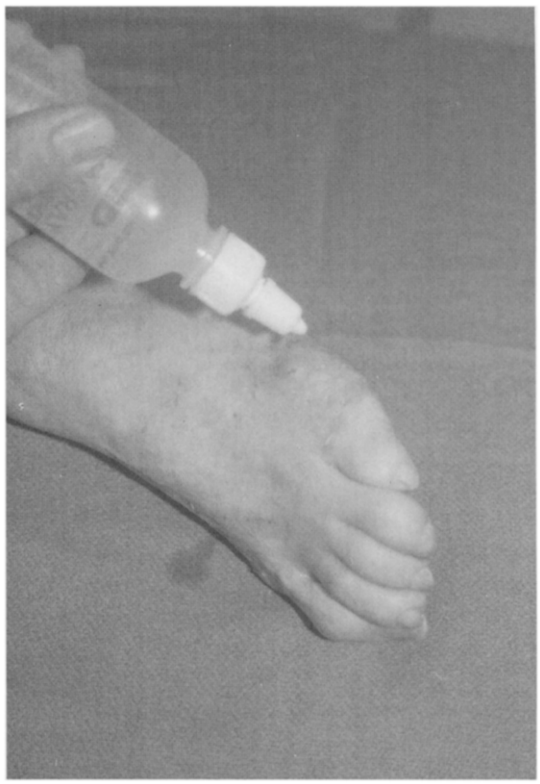

FIGURE 25 Application of Dermagran™ wound cleanser.

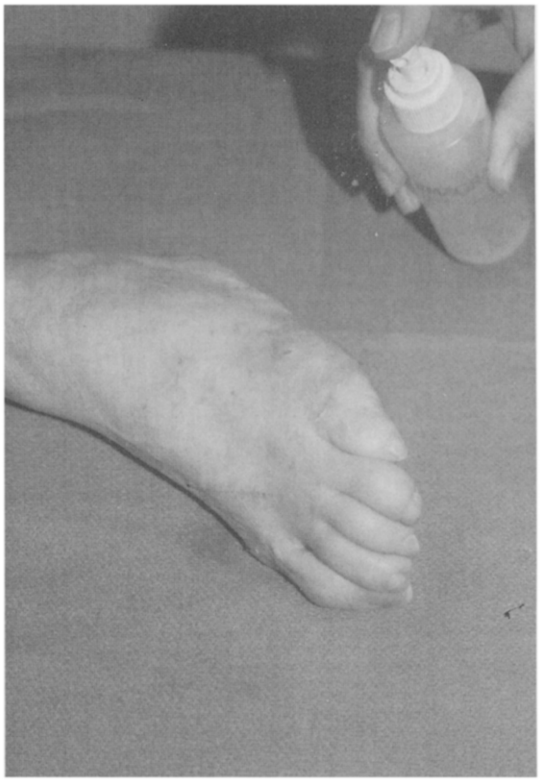

FIGURE 26 Application of Dermagran™ moisturizing spray.

TABLE 2 Classes of contact layer dressings

Class	Indication	Example
Transparent Film	Dry wounds—prevention desiccation, encourage moist environment	Bioclusive™ (Johnson & Johnson) POLYSKIN II™ (Kendall)
Hydrogels	Lightly exudative—nonadherent, encourage moist environment	NU-GEL™ (Johnson & Johnson)
Impregnated gauzes	Nonadherent (open mesh)—moderate to heavily exudative, prevent desiccation	Adaptic™ (Johnson & Johnson) Aquaphor™ (Beiersdorf)
	Nonadherent (fine mesh)—dry to lightly exudative, encourage moist environment	Petrolatum gauze U.S.P. (Sparta) Xeroform™ (Sparta)
	Contaminated/superficially infected wounds	Betadine™ Antiseptic Gauze (Purdue Frederick)
Nonadherent	Lightly exudative—absorbent, prevent maceration	Release™ (Johnson & Johnson) Telfa™ (Kendall)
Wet dressings	Dry, crusted, desiccated wounds	Sterile water wet dressing (Sparta) Isotonic saline wet dressing (Sparta)
	Draining, contaminated wounds	Hypertonic Wet Dressing (Sparta)
	Clean, dry wound—nonadherent	Owens Dressing (American Cyanamid)
Hydrocolloids	Lightly exudative wounds—provide occlusion and promotes moist environment	DuoDERM™ (Convatec)
	Good when autolytic debridement is desired	
Calcium alginate	Moderately to heavily exudative wounds—absorbent, prevent maceration	Sorbsan™ (Dow B. Hickam)
Exudate absorbers	Moderate to heavily exudative wounds—absorbent, enhance autolytic debridement	Debrisan™ (Johnson & Johnson)
Composite dressings	Various uses depending on composition of dressing	Mitraflex™ (Calgon Vestal)
Silicone gel	Hypertrophic scars and keloids—decrease size and reduce pain/itching	SILASTIC™ gel sheeting (Smith & Nephew United)
	Prophylactic treatment for known scar/keloid formers	
Wound sprays	Dry or granulating wounds—encourage granulation, maintain moist environment	Dermagran™ moisturizing spray (Derma Sciences)

of using SILASTIC™ gel sheeting[21] (SGS) for the treatment of scars and keloids (38). SGS is a crosslinked dimethyl and vinyl end blocked polydimethylsiloxane polymer. It is supplied in sheet form and reinforced with a fine polyester mesh (38, 39). The SGS is placed over an existing scar or keloid and held in place with adhesive tape. This can also be done on new surgical incisions of wounds closed by primary intention (after suture removal) in patients who are known keloid or hypertrophic scar formers (38). In the study cited, treatment lasted from 2 weeks to 2 months. An improvement in the appearance of scars and keloids (decrease in elevation, thickness and color) was seen in the vast majority of patients. Also seen was a decrease in abnormal sensation of the scar including pruritus, pain, and numbness (38) (Figs. 21–23).

Not falling into a dressing category, but nonetheless finding great use in wound healing, are various cleansing, lubricating, and stimulating sprays such as Dermagran™ moisturizing spray[22] and Dermagran™ wound cleanser.[23] They add moisture to wounds and stimulate the local circulation. Irrigation and cleansing of dry wounds can be accomplished prior to dressing application. They can also be used to help prevent disruption of fragile tissue at dressing changes (26). These products generally have an ingredient such as balsam or Peru that acts as a capillary stimulant (26). Zinc is often present, which is an important nutrient for healing wounds. Castor oil found in these sprays acts to soften dried tissue or eschar (26) (Figs. 24–26). The classes of contact layer dressings are listed in Table 2.

Summary

A review of the process of wound healing, along with influencing factors has been presented. The authors have defined and discussed the importance of the contact layer in surgical and wound dressings. A review of some common products used for this layer have been

[21] Smith & Nephew United Inc., Largo, FL 34649-1970.

[22,23] Derma Sciences, Old Forge, PA 18518.

presented. The importance of considering the type of wound and the conditions present when selecting one of these products has been stressed.

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